Letters to the Editor

More on right atrial mobile thrombi

In an issue of the journal, the problem of right atrial mobile thrombi (RAMT) was brought to the attention of readers by two renowned groups of cardiologists[1,2].

It is amazing how little has changed in our understanding of the optimal treatment of this dramatic clinical entity since the first systematic attempt to approach this problem a decade ago[3]. While the group from Aachen[1] supports thrombolytic therapy as the treatment of choice, available evidence favours the colleagues from Uxbridge[2], that existing options (heparin, thrombolysis and surgery) were similarly effective[4,6]. The strong and rather arbitrary criticism of the surgical approach to RAMT, expressed by the group from Aachen is in clear contrast with the recent report of Chapoutot *et al.*[6]. On the other hand, deaths were reported during thrombolytic therapy in RAMT, even among patients haemodynamically stable prior to its introduction[7].

Obviously the thrombus, if it is mobile and yet remains in the right atrium, *must be anchored somewhere*. In the majority of cases the shape of the thrombus leaves little doubt that we are dealing with a venous embolus in transit through the right heart chambers. Even if one is unable to visualize it, a fragment of RAMT is probably either caught in the patent foramen ovale or entangled in the chordae tendinae or Chiari network. Obviously this anchoring fragment is under mechanical strain and even without thrombolysis it does not hold for long, as evidenced by the high early fatal reembolization rate if RAMT are left untreated[3]. Thus, thrombolysis should be expected to increase the risk of dislodgement of RAMT or its fragments to the pulmonary arteries. It may be that aggressive front loading or bolus rt-PA regimens used by the Aachen group keeps pulmonary resistance low, due to rapid lysis of intrapulmonary thromboemboli. However, in view of the intra-cerebral haemorrhage in one of their four patients, this approach could not be considered superior to that reported earlier by others.

Unfortunately, data from randomized studies are neither available nor expected. This is probably why, despite the dramatic clinical picture and 2–5% prevalence among patients with pulmonary embolism, RAMT is not mentioned in the recent AHA Statement for Healthcare Professionals on Management of Deep Vein Thrombosis and Pulmonary Embolism. A few years ago, the RAMT Outcome Registry was created jointly by the members of the Working Groups on Pulmonary Circulation and Echocardiography of the European Society of Cardiology. The Registry aimed at prospective collection of homogeneous data regarding the appearance of right atrial mobile thrombi and also clinical and echocardiographic presentation and applied treatment. Since that time, over 70 cases have been reported to the Steering Committee. The design and early results, which seem to favour surgery, were presented at the recent Congress of the European Society of Cardiology, mostly with the aim of attracting the interest of additional centres. Though not ideal, this way of collecting data on RAMT may result in more evidence-based conclusions and recommendations than those from short single-centre series which continue to be both controversial and non-comparable.

The Registry is open to all interested centres. Information on the Registry and CRFs are available through the Working Group on Pulmonary Circulation and Right Ventricular Function FAX +48-22-6597506, E-mail: torbicki@amwaw.edu.pl. Please head your communication 'RAMT Outcome Registry'.

A. TORBICKI

Department of Hypertension and Angiology, Medical Academy, Warsaw, Poland

References

[1] Lepper W, Janssens U, Klues HG, Hanrath P. Successful lysis of mobile right heart and pulmonary artery thrombi, diagnosis and monitoring by transesophageal echocardiography (letter). Eur Heart J 1996; 17: 1603–4.

[2] Lo SSS, Sutton GC. Right atrial thrombus presenting in a patient with heart failure and disseminated intravascular coagulation (letter). Eur Heart J 1996; 17: 1605–6.

[3] Torbicki A, Pasierski T, Uchman Z, Miskiewicz Z. Right atrial mobile thrombi: two-dimensional echocardiographic diagnosis and clinical outcome. Cor Vas 1987; 29: 293–303.

[4] Kinney EL, Wright RJ. Efficacy of treatment of patients with echocardiographically detected right-sided heart thrombi. A meta-analysis. Am Heart J 1989; 118: 569–73.

[5] Kronik G. European Cooperative Study on the clinical significance of right heart thrombi. Eur Heart J 1989; 10: 1046–59.

[6] Chapoutot L, Nazeyrollas P, Metz D *et al.* Floating right heart thrombi and pulmonary embolism: diagnosis, outcome and therapeutic management. Cardiology 1996; 87: 169–74.

[7] Torbicki A, Kronik G, Tramarin R *et al.* Therapeutic preferences and outcome of various treatments of venous clots trapped in the right atrium. Preliminary results of a European Registry. Eur Heart J 1996; 17 (Abstr Suppl). 244.

Coronary velocity pressure tracings

Drs Di Mario and Serruys are to be congratulated on their comprehensive overview on principles of coronary velocity and pressure tracings published in a supplement to the journal in 1995[1]. They address the important issue of why and how functional, and not just structural, aspects of coronary artery stenoses severity have to be assessed in order to provide a sound basis for therapeutic decisions, especially in cases of intermediate lesions. In particular, they provide the reader with valuable theoretical information on the rationale for the application of coronary flow velocity indices in functional assessment. Thus, it becomes implicitly evident that the notion that the mean coronary artery flow velocity is constant throughout the epicardial coronary artery tree[2] must be an artefact which is probably related to insufficient measurement techniques.

On the other hand, their explanation for the use of proximal-to-distal velocity ratios may create some confusion as to why coronary artery flow velocity must decrease from proximal to distal. The reasons for this impression mainly relate to their Figure 4 and the accompanying legend and are as follows:

(1) Of minor importance, the reader of this article would probably profit from finding out what exactly is meant by the 'principle of limited/adaptive vascular shear stress' governing the size ratios of the arteries proximal and

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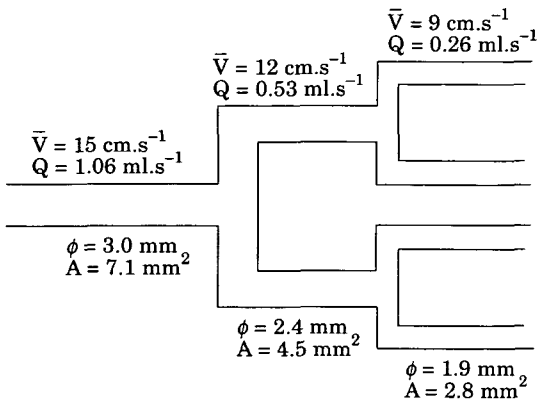

$A_M = 2^{2/3} \times \bar{A}_D$
$A_M = \bar{A}_D\ 1.588$

Total cross-sectional area increase per bifurcation = 1.26
Proximal to distal velocity ratio per bifurcation = 1.25

Figure 4 **Diagram showing a schematic variation of vascular diameter (φ, in mm), cross-sectional area (CSA, in mm^2), flow (Q, in ml . s^{-1}) and mean flow velocity ($\bar{V}$, in cm . s^{-1}) from proximal to distal in coronary arteries. In the example, the division between the mother artery (A_M) and the daughter artery is symmetrical and obeys the principle of minimum viscous energy loss and of limited/adaptive vascular shear stress[3,4]. According to this law, there is a decrease in the *average* cross-sectional area of the vessel ($\bar{A}_D$) distal to the bifurcation of $2^{2/3}$=1·588 which translates into an increase in total vascular area per bifurcation of 1·26. Note that the decrease in flow velocity from proximal to distal is approximately equal to the increase in *total* cross-sectional area per bifurcation which is in accordance with the law of mass conservation.**

distal to a bifurcation. It is not only the aforementioned principle but also the law of minimum energy loss for the transport of blood through the coronary arterial tree which governs the vascular caliber downstream relative to that upstream of a bifurcation[3,4].

(2) More importantly, the use of the terms 'area increase per bifurcation' and 'decrease in CSA; (figure legend) may puzzle the reader. It should be confirmed whether it is the sum or the average of the two vessels distal of a bifurcation. The same is true for the fact that no explanation is provided to indicate that $\bar{A}_D$ means the average cross-sectional area of the two daughter vessels.

(3) Finally, in the last sentence of the legend to Figure 4 ('note that the decrease in flow velocity from proximal to distal is much smaller than the decrease in cross-sectional area and volume flow') it appears that the authors have misconstrued the purpose of describing the coronary artery tree as a symmetrical structure. It is not in order to demonstrate that the flow velocity towards the vascular periphery decreases less than does the cross-sectional area of each *single* vessel distal to a bifircation. Rather, it is to illustrate more easily that the *decrease* in flow velocity (=1·25 according to Fig. 4) going downstream is equal to the *increase* in total cross-sectional area of *both* daughter vessels beyond each bifurcation (=1·26 according to Fig. 4), which is in accordance with the law of conservation of mass. Therefore, Fig. 4 and the legend to this figure are given again.

C. SEILER

Division of Cardiology, University Hospital Bern, Bern, Switzerland

References

[1] Di Mario C, Serruys P. Principles of interpretation of coronary velocity and pressure tracings. Eur Heart J 1995; 16 (Suppl J): 53–9.

[2] Ofili E, Kern M, Labovitz A. Analysis of coronary blood flow velocity dynamics in angiographically normal and stenoses arteries before and after endolumen enlargement by angioplasty. J Am Coll Cardiol 1993; 21: 308–16.

[3] Seiler C, Kirkeeide RL, Gould KL. Basic structure-function relations of the epicardial coronary vascular tree; Basis of quantitative coronary arteriography for diffuse coronary artery disease. Circulation 1992; 85: 1987–2003.

[4] Seiler C, Kirkeeide RL, Gould KL. Measurement from arteriograms of regional myocardial bed size distal to any point in the coronary vascular tree for assessing anatomic area at risk. J Am Coll Cardiol 1993; 21: 783–797.

A reply

Thank you for your appreciation of our efforts to illustrate the rationale of the different indices of stenosis severity based on intracoronary Doppler measurements.

Figure 4 was largely based on the findings of your experimental and clinical work, quoted as references [3] and [4] in your letter. As you suggest, the reader will certainly benefit from direct knowledge of these articles which, although not reporting intracoronary velocity measurements, explain why and how much flow velocity decreases along the arterial coronary tree. To convey this rather complex physiological concept, we thought that a scheme was easier to understand and we appreciate your criticisms and suggestions to make the drawing and legends more clear.

At the time this manuscript was written (December 1993) the proximal-to-distal velocity ratio was claimed to be the most specific velocity parameter of stenosis severity[1]. The anatomical variations in the three main coronary arteries (multiple bifurcations in the left anterior descending coronary artery, fewer, occasionally very large branches originating often very proximal from the left circumflex artery, no major branching points along the trunk of the right coronary artery) greatly affect the usefulness of this index when used in a large unselected patient population. The results obtained in 300 patients in the multicentre trial DEBATE have shown that the proximal-to-distal velocity ratio has no correlation with angiographic measurements of stenosis severity before angioplasty and is not predictive of the short- and long-term clinical outcome after PTCA[2].In clinical